

=> d his

(FILE 'HOME' ENTERED AT 10:51:48 ON 24 APR 2002)

FILE 'HCAPLUS' ENTERED AT 10:52:32 ON 24 APR 2002

L1 65 S LAHIRI J?/AU
 L2 2088 S FANG Y?/AU
 L3 122 S JONAS S?/AU
 L4 19 S KALAL P?/AU
 L5 10759 S WANG W?/AU
 L6 13029 S L1-5
 L7 526 S L6 AND ?MEMBRANE?
 L8 27 S L7 AND ASSAY?
 L9 3 S L8 AND (CHIP OR ?ARRAY? OR SURFACE OR ?SILAN? OR GLASS)
 L10 24 S L8 NOT L9
 L11 1 S L10 AND LIGAND(2A)BIND?
 L12 4 S L9 OR L11
 SELECT RN L12 1-4

- invento
search

FILE 'REGISTRY' ENTERED AT 10:59:10 ON 24 APR 2002

L13 4 S E1-4

FILE 'HCAPLUS' ENTERED AT 10:59:32 ON 24 APR 2002

L14 2 S L12 AND L13
 L15 4 S L12 OR L14 *4 citations for inventors*
 L16 509 S (?PROTEIN? OR ?PEPTID?) (2A)CHIP
 L17 1 S L16 AND L6
 L18 24061 S (?PROTEIN? OR ?PEPTID?) (5A) (?IMMOB? OR ATTACH? OR SPAN? OR FI
 L19 18 S L6 AND L18
 L20 9 S L19 AND (?ARRAY? OR SURFACE OR ?SILAN? OR GLASS OR ?MEMBRAN?)
 L21 9 S L20 NOT L15
 L22 10 S L17 OR L21 *10 cites for inventors*


FILE 'REGISTRY' ENTERED AT 11:15:25 ON 24 APR 2002

L23 141484 S "SILANE"
 L24 25828 S "AMINOPROPYL"
 L25 569 S L23 AND L24
 L26 220 S L25 AND "GAMMA"
 L27 57 S L26 AND NC=1 NOT PMS/CI
 L28 43 S L27 NOT RSD/FA
 L29 3 S L28 NOT O/ELS
 L30 1 S L29 AND C3 H11 N SI/MF *claim 51 cpd*
 L31 0 S 6382-82-7 /CRN

FILE 'HCAPLUS' ENTERED AT 11:21:53 ON 24 APR 2002

L32 3 S L30/PREP *3 cites for prep. of 8-aminopropylsilane*

FILE 'CASREACT' ENTERED AT 11:25:07 ON 24 APR 2002

L33 STR 6382-82-7
 L34 0 S L33
 L35 0 S L33 FUL *no cites for prep of* 

FILE 'HCAPLUS' ENTERED AT 12:24:50 ON 24 APR 2002

L36 156729 S L16 OR L18 OR (?PROTEIN? OR ?PEPTID?) (5A) (?ASSOCIAT? OR ?COU
 L37 668665 S ?MEMBRAN? OR ?BILAYER? OR ?AMPHIPHILIC? (3A) ?SURFAC? OR ?PROT
 L38 289077 S ?PRINT? OR QUILL? OR QUILL-PIN OR SPOT? OR MICROSPOT?
 L39 1041533 S GLASS OR SILICA OR QUARTZ
 L40 73 S L30
 L41 43272 S G-PROTEIN
 L42 349 S L36 AND L37 AND L38

L43 1 S L42 AND L40
 E HIW
 L44 1 S L39 AND L43
 L45 1 S L44 AND L41 *1 cite*
 L46 14 S L42 AND L39
 L47 2 S L46 AND L41
 L48 1 S L47 NOT (L45 OR L12) *1 cite*
 L49 12 S L46 NOT L47-48
 L50 5 S L49 AND PATENT/DT
 L51 4 S L50 AND PRD<20000810
 L52 7 S L49 NOT L50
 L53 5 S L52 AND PD<20000810
 L54 9 S L51 OR L53 *9 cites*
 L55 11 S L39 AND L38 AND L41
 L56 9 S L55 NOT (L46 OR L12 OR L48)
 L57 3 S L56 AND L36-37 *3 cites*
 L58 349 S L36 AND L37 AND L38
 L59 5 S L58 AND CHIP
 L60 16 S L58 AND MICROARRAY
 L61 19 S L59-60
 L62 4 S L61 AND L41
 L63 2 S L62 NOT (L46 OR L12 OR L48) *2 cites*
 L64 4 S L61 AND L39
 L65 3 S L64 NOT L62-63
 L66 2 S L65 AND PRD<20000810 *2 patents*
 L67 2 S L40 AND L38
 L68 4 S L40 AND (L38 OR L41 OR L36-38)
 L69 3 S L68 NOT (L46 OR L12 OR L48) *3 cites*
 L70 9141 S L36(P)ASSAY?
 L71 841989 S PROBE OR TARGET OR CONTACT
 L72 1244 S L70 AND L71
 L73 295 S L70 AND (CONTACT? OR SPOT? OR MICROSPOT?)
 L74 87 S L73 AND DETECT?
 L75 12 S L74 AND TARGET
 L76 9 S L74 AND PROBE
 L77 4 S L75 AND L76
 L78 16 S L74 AND SOLUTION
 L79 2 S L77 AND L78 *2 sites*
 L80 30123 S L37 (P)ASSAY?
 L81 2210 S L70 AND L80
 L82 2170 S L70 (P)L80
 L83 63 S L82(P) (CONTACT? OR SPOT? OR MICROSPOT? OR PIN)
 L84 6 S L83(P) (TARGET OR PROBE)
 L85 0 S L83 AND (L39 OR SIO2)
 L86 13 S L82 AND (L39 OR SIO2)
 L87 0 S L86 AND (CONTACT? OR SPOT? OR MICROSPOT? OR PIN OR TIP)
 L88 80 S L81 AND (CONTACT? OR SPOT? OR MICROSPOT? OR PIN OR TIP)
 L89 2 S L88 AND (SIO2 OR L39)
 L90 19 S L88 AND SURFACE
 L91 477 S CHIP(P)ASSAY?
 L92 126 S L91(P)SURFACE
 L93 11 S L92(P)?MEMBRANE? *11 cites*
 L94 18 S (PROTEIN OR ?PEPTID?) (5A)CHIP(10A)PREPAR?
 L95 0 S L94 AND PIN
 L96 4 S L94 AND ?MEMBRAN?
 L97 0 S L94 AND BLOT?
 L98 1 S L94 AND (?PATTERN? OR ?SPOT?)
 L99 4 S L96 OR L98
 L100 3 S L99 NOT (L46 OR L12 OR L48) *3 cites*

Invent Search

TRAN 09/854,786

=> d que 115

L1	65	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	LAHIRI J?/AU
L2	2088	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	FANG Y?/AU
L3	122	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	JONAS S?/AU
L4	19	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	KALAL P?/AU
L5	10759	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	WANG W?/AU
L6	13029	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	(L1 OR L2 OR L3 OR L4 OR L5)
L7	526	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	L6 AND ?MEMBRANE?
L8	27	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	L7 AND ASSAY?
L9	3	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	L8 AND (CHIP OR ?ARRAY? OR SURFACE OR ?SILAN? OR GLASS)
L10	24	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	L8 NOT L9
L11	1	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	L10 AND LIGAND(2A)BIND?
L12	4	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	L9 OR L11
L13	4	SEA	FILE=REGISTRY	ABB=ON	PLU=ON	(127361-24-4/BI OR 217962-21- 5/BI OR 250154-17-7/BI OR 80451-05-4/BI)
L14	2	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	L12 AND L13
L15	4	SEA	FILE=HCAPLUS	ABB=ON	PLU=ON	L12 OR L14

4 cites

=> d ibib abs hitstr 1

L15 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2002:145010 HCAPLUS

TITLE: **Membrane Protein Microarrays**AUTHOR(S): **Fang, Ye; Frutos, Anthony G.; Lahiri, Joydeep**

CORPORATE SOURCE: Biochemical Technologies, Science and Technology Division, Corning Incorporated, Corning, NY, 14831, USA

SOURCE: Journal of the American Chemical Society (2002), 124(11), 2394-2395

CODEN: JACSAT; ISSN: 0002-7863

PUBLISHER: American Chemical Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB This paper describes the fabrication of **microarrays** consisting of G protein-coupled receptors (GPCRs) on **surfaces** coated with .gamma.-**aminopropylsilane** (GAPS). Microspots of model **membranes** on GAPS-coated **surfaces** were obsd. to have several desired properties-high mech. stability, long range lateral fluidity, and a thickness corresponding to a lipid bilayer in the bulk of the microspot. GPCR **arrays** were obtained by printing **membrane** preps. contg. GPCRs using a quill-pin printer. To demonstrate specific binding of ligands, **arrays** presenting neurotensin (NTR1), adrenergic (.beta.1), and dopamine (D1) receptors were treated with fluorescently labeled neurotensin (BT-NT). Fluorescence images revealed binding only to microspots corresponding to the neurotensin receptor; this specificity was further demonstrated by the inhibition of binding in the presence of excess unlabeled neurotensin. The ability of GPCR **arrays** to enable selectivity studies between the different subtypes of a receptor was examd. by printing **arrays** consisting of three subtypes of the adrenergic receptor: .beta.1, .beta.2, and .alpha.2A. When treated with fluorescently labeled CGP 12177, a cognate antagonist analog specific to .beta.-adrenergic receptors, binding was only obsd. to microspots of the .beta.1 and .beta.2 receptors. Furthermore, binding of labeled CGP 12177 was inhibited when the **arrays** were incubated with solns. also contg. ICI 118551, and in a manner consistent with the higher affinity of ICI 118551 for the .beta.2 receptor relative to that for the .beta.1 receptor. The ability to est. binding affinities of compds. using GPCR **arrays** was examd. using a competitive binding **assay** with BT-NT and unlabeled neurotensin on NTR1 **arrays**. The estd. IC50 value (2 nM) for neurotensin is in agreement with the literature; this agreement suggests that the receptor-G protein complex is preserved in the microspot. This first ever demonstration of direct pin-printing of **membrane** proteins and ligand-binding **assays** thereof fills a significant void in protein microchip technol.-the lack of practical **microarray**-based methods for **membrane** proteins.

REFERENCE COUNT: 18 THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TRAN 09/854,786

=> d ind 1

L15 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2002 ACS
CC 9 (Biochemical Methods)

=> d ibib abs hitstr 2

L15 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:230792 HCAPLUS

DOCUMENT NUMBER: 134:363049

TITLE: Kinetics of **membrane** lysis by custom lytic peptides and peptide orientations in **membrane**

AUTHOR(S): Chen, H. M.; Clayton, A. H. A.; Wang, W.; Sawyer, W. H.

CORPORATE SOURCE: Department of Biochemistry, Hong Kong University of Science and Technology, Kowloon, Hong Kong

SOURCE: European Journal of Biochemistry (2001), 268(6), 1659-1669

CODEN: EJBCAI; ISSN: 0014-2956

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB To aid the development of custom peptide antibiotics, a kinetic study of **membrane** lysis by cecropin B (CB) and its analogs, cecropin B1 (CB1) and cecropin B3 (CB3) was carried out to det. the mechanism by which these peptides disrupt the bilayer structure of liposomes of defined compn. Disruption of the phospholipid bilayer was detd. by a fluorescence **assay** involving the use of dithionite to quench the fluorescence of lipids labeled with N-7-nitro-2,1,3-benzoxadiazol-4-yl. Lytic peptides caused the disruption of liposomes to occur in two kinetic steps. For liposomes composed of mixts. of phosphatidylcholine and phosphatidic acid, the time consts. for each kinetic step were shorter for CB and CB1 than for CB3. Oriented CD expts. showed that the peptides could exist in at least two different **membrane**-assocd. states that differed primarily in the orientation of the helical segments with respect to the bilayer **surface**. The results are discussed in terms of kinetic mechanisms of **membrane** lysis. The mode of actions of these peptides used for the interpretation of their kinetic mechanisms were supported by **surface** plasmon resonance expts. including or excluding the pore-forming activities.

IT 80451-05-4, Cecropin B 127361-24-4, Cecropin B1

217962-21-5, Cecropin B3

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study) (kinetics of **membrane** lysis by custom lytic peptides and peptide orientations in **membrane** using fluorescence-quenching and oriented CD spectroscopy)

RN 80451-05-4 HCAPLUS

CN Cecropin B (Platysamia cecropia antibacterial peptide) (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

RN 127361-24-4 HCAPLUS

CN L-Lysinamide, L-lysyl-L-tryptophyl-L-lysyl-L-valyl-L-phenylalanyl-L-lysyl-L-lysyl-L-isoleucyl-L-.alpha.-glutamyl-L-lysyl-L-methionylglycyl-L-arginyl-L-asparaginyl-L-isoleucyl-L-arginyl-L-asparaginylglycyl-L-isoleucyl-L-valyl-L-lysyl-L-alanyl-glycyl-L-prolyl-L-lysyl-L-tryptophyl-L-lysyl-L-valyl-L-phenylalanyl-L-lysyl-L-lysyl-L-isoleucyl-L-.alpha.-glutamyl- (9CI) (CA INDEX NAME)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

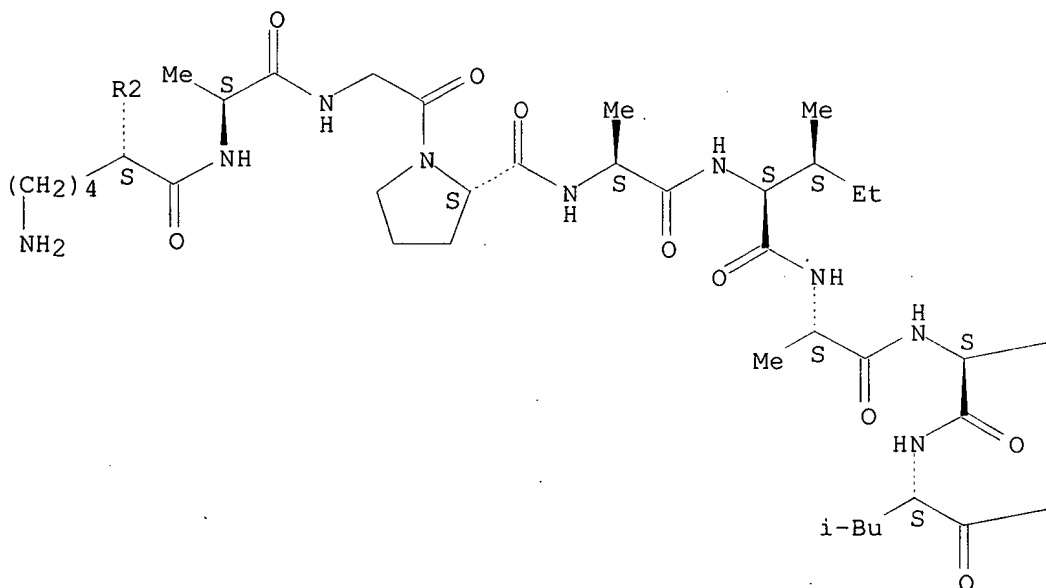
RN 217962-21-5 HCAPLUS

CN L-Leucinamide, L-alanyl-L-isoleucyl-L-alanyl-L-valyl-L-leucylglycyl-L-.alpha.-glutamyl-L-alanyl-L-lysyl-L-alanyl-L-leucyl-L-methionylglycyl-L-

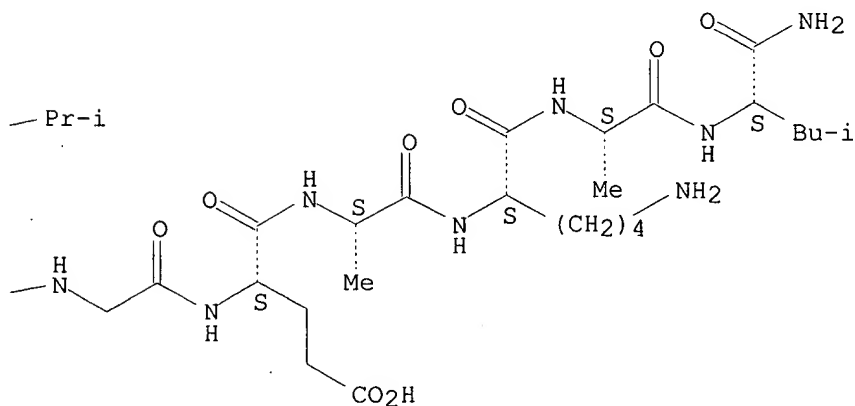
arginyl-L-asparaginyl-L-isoleucyl-L-arginyl-L-asparaginylglycyl-L-isoleucyl-L-valyl-L-lysyl-L-alanylglycyl-L-prolyl-L-alanyl-L-isoleucyl-L-alanyl-L-valyl-L-leucylglycyl-L-.alpha.-glutamyl-L-alanyl-L-lysyl-L-alanyl-
(9CI) (CA INDEX NAME)

Absolute stereochemistry.

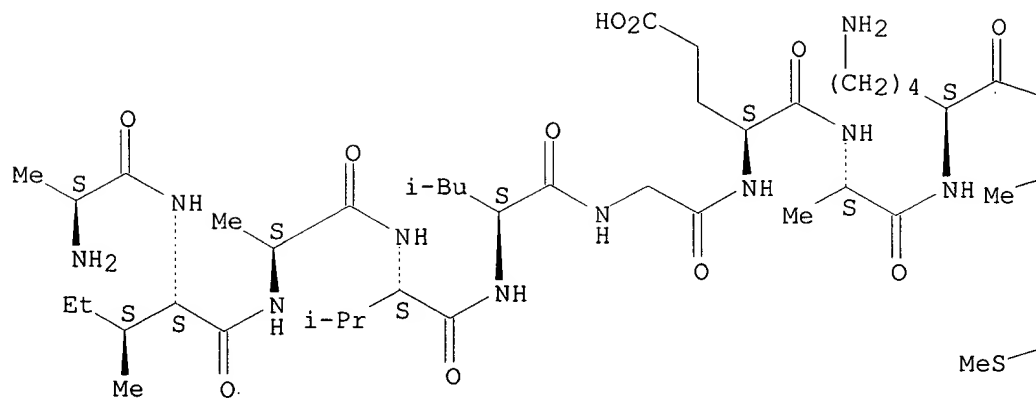
PAGE 1-A



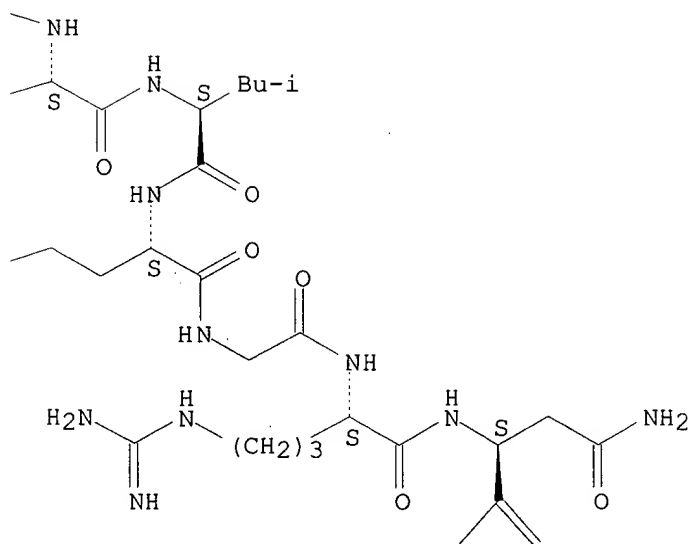
PAGE 1-B

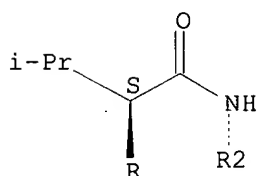
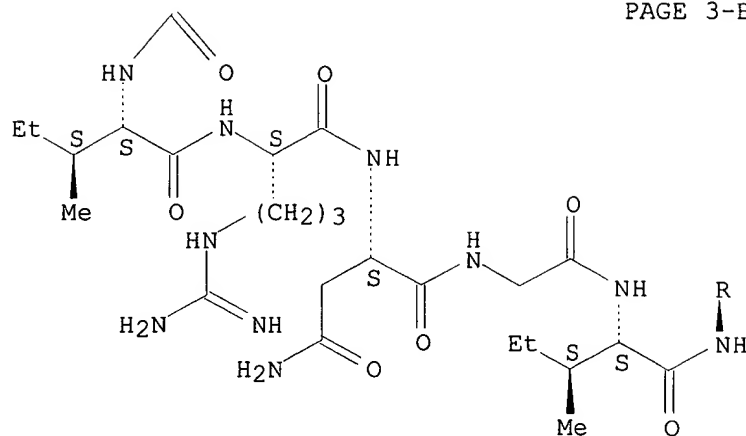


PAGE 2-A



PAGE 2-B





REFERENCE COUNT:

31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ind 2

L15 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2002 ACS

CC 6-6 (General Biochemistry)

ST kinetics **membrane** lysis peptide

IT **Membrane**, biological

(bilayer; kinetics of **membrane** lysis by custom lytic peptides and peptide orientations in **membrane** using fluorescence-quenching and oriented CD spectroscopy)

IT Physical process kinetics

(**membrane** lysis; kinetics of **membrane** lysis by custom lytic peptides and peptide orientations in **membrane** using fluorescence-quenching and oriented CD spectroscopy)

IT Phosphatidic acids

Phosphatidylcholines, biological studies

Phospholipids, biological studies

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(**membrane**; kinetics of **membrane** lysis by custom lytic peptides and peptide orientations in **membrane** using fluorescence-quenching and oriented CD spectroscopy)

IT Conformation

(protein; kinetics of **membrane** lysis by custom lytic peptides and peptide orientations in **membrane** using fluorescence-quenching and oriented CD spectroscopy)

IT 80451-05-4, Cecropin B 127361-24-4, Cecropin B1

217962-21-5, Cecropin B3

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)

(kinetics of **membrane** lysis by custom lytic peptides and peptide orientations in **membrane** using fluorescence-quenching and oriented CD spectroscopy)

=> d ibib abs hitstr 3

L15 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:5303 HCAPLUS

DOCUMENT NUMBER: 132:133833

TITLE: Affinity purification and partial characterization of the zonulin/zonula occludens toxin (Zot) receptor from human brain

AUTHOR(S): Lu, R.; Wang, W.; Uzzau, S.; Vigorito, R.; Zielke, H. R.; Fasano, A.

CORPORATE SOURCE: Division of Pediatric Gastroenterology and Nutrition and Center for Vaccine Development, University of Maryland School of Medicine, Baltimore, MD, 21201, USA

SOURCE: Journal of Neurochemistry (2000), 74(1), 320-326

CODEN: JONRA9; ISSN: 0022-3042

PUBLISHER: Lippincott Williams & Wilkins

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The intercellular tight junctions (TJs) of endothelial cells represent the limiting structure for the permeability of the blood-brain barrier (BBB). Although the BBB has been recognized as being the interface between the bloodstream and the brain, little is known about its regulation. Zonulin and its prokaryotic analog, zonula occludens toxin (Zot) elaborated by *Vibrio cholerae*, both modulate intercellular TJs by binding to a sp. **surface** receptor with subsequent activation of an intracellular signaling pathway involving phospholipase C and protein kinase C activation and actin polymn. Affinity column purifn. revealed that human brain plasma **membrane** preps. contain two Zot binding proteins of .apprx.55 and .apprx.45 kDa. Structural and kinetic studies, including satn. and competitive **assays**, identified the 55-kDa protein as tubulin, whereas the 45-kDa protein represents the zonulin/Zot receptor. Biochem. characterization provided evidence that this receptor is a glycoprotein contg. multiple sialic acid residues. Comparison of the N-terminal sequence of the zonulin/Zot receptor with other protein sequences by BLAST anal. revealed a striking similarity with MRP-8, a 14-kDa member of the S-100 family of calcium binding proteins. The discovery and characterization of this receptor from human brain may significantly contribute to our knowledge on the pathophysiol. regulation of the BBB.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ind 3

L15 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2002 ACS

CC 6-3 (General Biochemistry)

Section cross-reference(s): 4, 13

ST zonulin zonula occludens toxin receptor protein brain human

IT Proteins, specific or class

RL: BSU (Biological study, unclassified); BIOL (Biological study)

(MRP-8 (migration-inhibiting factor-related, 8000-mol.-wt.); affinity purifn. and partial characterization of zonulin/zonula occludens toxin (Zot) receptor from human brain)

IT Proteins, specific or class

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(Zot binding protein; affinity purifn. and partial characterization of zonulin/zonula occludens toxin (Zot) receptor from human brain)

IT Blood-brain barrier

Brain

Heart

Intestine

Protein sequences

(affinity purifn. and partial characterization of zonulin/zonula occludens toxin (Zot) receptor from human brain)

IT Cell junction

(tight junction; affinity purifn. and partial characterization of zonulin/zonula occludens toxin (Zot) receptor from human brain)

IT Receptors

RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)

(zonulin/zonula occludens toxin; affinity purifn. and partial characterization of zonulin/zonula occludens toxin (Zot) receptor from human brain)

IT Toxins

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(zonulin/zonula occludens toxin; affinity purifn. and partial characterization of zonulin/zonula occludens toxin (Zot) receptor from human brain)

=> d ibib abs hitstr 4

L15 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:651397 HCAPLUS

DOCUMENT NUMBER: 131:333706

TITLE: Identification of a talin-binding site in the integrin .beta.3 subunit distinct from the NPLY regulatory motif of post-ligand binding functions. The talin N-terminal head domain interacts with the **membrane**-proximal region of the .beta.3 cytoplasmic tail

AUTHOR(S): Patil, Sonali; Jedsadayanmata, Arom; Wencel-Drake, June D.; Wang, Wei; Knezevic, Irina; Lam, Stephen C.-T.

CORPORATE SOURCE: Department of Pharmacology, The University of Illinois, Chicago, IL, 60612, USA

SOURCE: J. Biol. Chem. (1999), 274(40), 28575-28583

CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Following platelet aggregation, integrin .alpha.IIb.beta.3 becomes assocd. with the platelet cytoskeleton. The conserved NPLY sequence represents a potential .beta.-turn motif in the .beta.3 cytoplasmic tail and has been suggested to mediate the interaction of .beta.3 integrins with talin. In the present study, the authors performed a double mutation (N744Q/P745A) in the integrin .beta.3 subunit to test the functional significance of this .beta.-turn motif. Chinese hamster ovary cells were co-transfected with cDNA constructs encoding mutant .beta.3 and wild type .alpha.IIb. Cells expressing either wild type (A5) or mutant (D4) .alpha.IIb.beta.3 adhered to fibrinogen; however, as opposed to control A5 cells, adherent D4 cells failed to spread, form focal adhesions, or initiate protein tyrosine phosphorylation. To investigate the role of the NPLY motif in talin binding, the authors examd. the ability of the mutant .alpha.IIb.beta.3 to interact with talin in a solid phase binding **assay**. Both wild type and mutant .alpha.IIb.beta.3, purified by RGD affinity chromatog., bound to a similar extent to immobilized talin. Addnl., purified talin failed to interact with peptides contg. the AKWDTANNPLYK sequence indicating that the talin binding domain in the integrin .beta.3 subunit does not reside in the NPLY motif. In contrast, specific binding of talin to peptides contg. the **membrane**-proximal HDRKEFAKFEEERARAK sequence of the .beta.3 cytoplasmic tail was obsd., and this interactions was blocked by a recombinant protein fragment corresponding to the 47-kDa N-terminal head domain of talin (rTalin-N). In addn., RGD affinity purified platelet .alpha.IIb.beta.3 bound dose-dependently to immobilized rTalin-N, indicating that an integrin-binding site is present in the talin N-terminal head domain. Collectively, these studies demonstrate that the NPLY .beta.-turn motif regulates post-ligand binding functions of .alpha.IIb.beta.3 in a manner independent of talin interaction. Moreover, talin was shown to bind through its N-terminal head domain to the **membrane**-proximal sequence of the .beta.3 cytoplasmic tail.

IT 250154-17-7

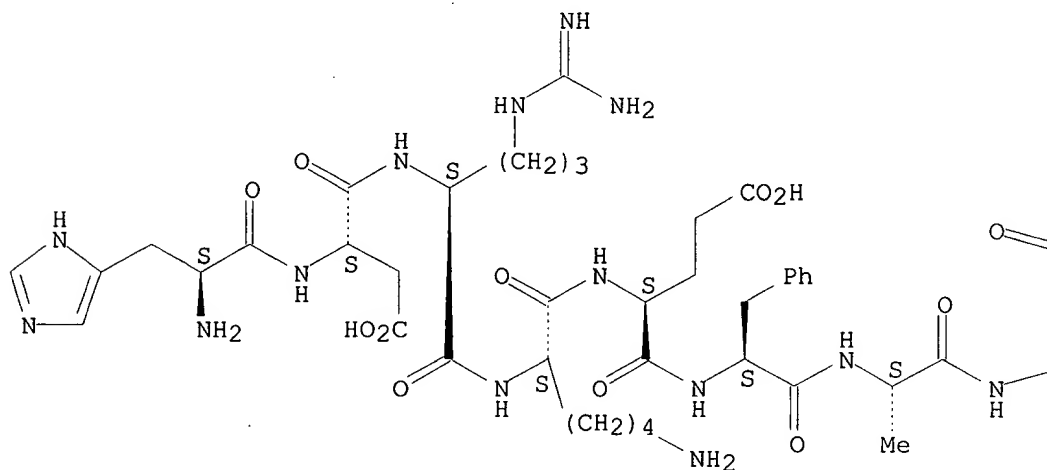
RL: BOC (Biological occurrence); BPR (Biological process); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process) (as talin N-terminal head domain-binding site in integrin .beta.3 subunit **membrane**-proximal cytoplasmic tail region)

RN 250154-17-7 HCAPLUS

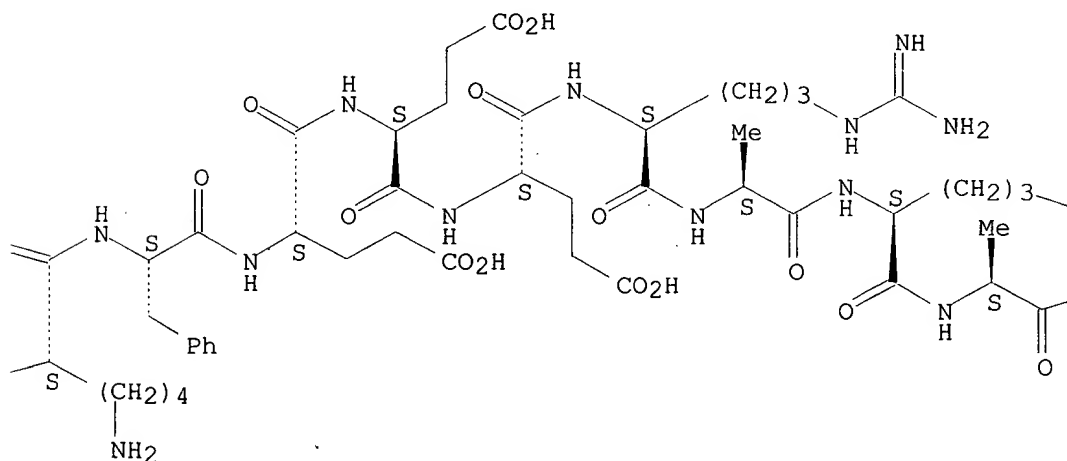
CN L-Lysine, L-histidyl-L-.alpha.-aspartyl-L-arginyl-L-lysyl-L-.alpha.-glutamyl-L-phenylalanyl-L-alanyl-L-lysyl-L-phenylalanyl-L-.alpha.-glutamyl-L-.alpha.-glutamyl-L-.alpha.-glutamyl-L-arginyl-L-alanyl-L-arginyl-L-alanyl- (9CI) (CA INDEX NAME)

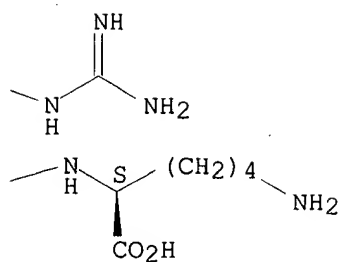
Absolute stereochemistry.

PAGE 1-A



PAGE 1-B





REFERENCE COUNT:

60

THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ind 4

L15 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2002 ACS
 CC 6-3 (General Biochemistry)
 ST talin N terminal head domain binding site integrin beta3
 IT Fibrinogens
 RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
 (adhesion to; talin N-terminal head domain-binding site in integrin
 .beta.3 subunit **membrane**-proximal cytoplasmic tail region
 distinct from NPLY .beta.-turn regulatory motif of post-ligand
binding functions in relation to)
 IT Platelet (blood)
 (aggregation; talin N-terminal head domain-binding site in integrin
 .beta.3 subunit **membrane**-proximal cytoplasmic tail region
 distinct from NPLY .beta.-turn regulatory motif of post-ligand
binding functions in relation to)
 IT Cell junction
 (focal contact; talin N-terminal head domain-binding site in integrin
 .beta.3 subunit **membrane**-proximal cytoplasmic tail region
 distinct from NPLY .beta.-turn regulatory motif of post-ligand
binding functions in relation to)
 IT Cell aggregation
 (platelet; talin N-terminal head domain-binding site in integrin
 .beta.3 subunit **membrane**-proximal cytoplasmic tail region
 distinct from NPLY .beta.-turn regulatory motif of post-ligand
binding functions in relation to)
 IT Phosphorylation, biological
 (protein; talin N-terminal head domain-binding site in integrin .beta.3
 subunit **membrane**-proximal cytoplasmic tail region distinct
 from NPLY .beta.-turn regulatory motif of post-ligand
binding functions in relation to)
 IT Protein motifs
 (talin N-terminal head domain-binding site in integrin .beta.3 subunit
membrane-proximal cytoplasmic tail region distinct from NPLY
 .beta.-turn regulatory motif of post-ligand **binding**
 functions)
 IT Talin
 RL: BPR (Biological process); PRP (Properties); BIOL (Biological study);
 PROC (Process)
 (talin N-terminal head domain-binding site in integrin .beta.3 subunit
membrane-proximal cytoplasmic tail region distinct from NPLY
 .beta.-turn regulatory motif of post-ligand **binding**
 functions)
 IT Integrins
 RL: BPR (Biological process); PRP (Properties); BIOL (Biological study);
 PROC (Process)
 (.alpha.IIb.beta.3; talin N-terminal head domain-binding site in
 integrin .beta.3 subunit **membrane**-proximal cytoplasmic tail
 region distinct from NPLY .beta.-turn regulatory motif of post-
ligand binding functions)
 IT Integrins
 RL: BPR (Biological process); PRP (Properties); BIOL (Biological study);
 PROC (Process)
 (.beta.3; talin N-terminal head domain-binding site in integrin .beta.3
 subunit **membrane**-proximal cytoplasmic tail region distinct
 from NPLY .beta.-turn regulatory motif of post-ligand
binding functions)
 IT 250154-17-7
 RL: BOC (Biological occurrence); BPR (Biological process); PRP

TRAN 09/854,786

(Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(as talin N-terminal head domain-binding site in integrin .beta.3
subunit **membrane**-proximal cytoplasmic tail region)

*Inventor she
search*

TRAN 09/854,786

=> d que 122

L1 65 SEA FILE=HCAPLUS ABB=ON PLU=ON LAHIRI J?/AU
L2 2088 SEA FILE=HCAPLUS ABB=ON PLU=ON FANG Y?/AU
L3 122 SEA FILE=HCAPLUS ABB=ON PLU=ON JONAS S?/AU
L4 19 SEA FILE=HCAPLUS ABB=ON PLU=ON KALAL P?/AU
L5 10759 SEA FILE=HCAPLUS ABB=ON PLU=ON WANG W?/AU
L6 13029 SEA FILE=HCAPLUS ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4 OR L5)
L7 526 SEA FILE=HCAPLUS ABB=ON PLU=ON L6 AND ?MEMBRANE?
L8 27 SEA FILE=HCAPLUS ABB=ON PLU=ON L7 AND ASSAY?
L9 3 SEA FILE=HCAPLUS ABB=ON PLU=ON L8 AND (CHIP OR ?ARRAY? OR
SURFACE OR ?SILAN? OR GLASS)
L10 24 SEA FILE=HCAPLUS ABB=ON PLU=ON L8 NOT L9
L11 1 SEA FILE=HCAPLUS ABB=ON PLU=ON L10 AND LIGAND(2A)BIND?
L12 4 SEA FILE=HCAPLUS ABB=ON PLU=ON L9 OR L11
L13 4 SEA FILE=REGISTRY ABB=ON PLU=ON (127361-24-4/BI OR 217962-21-
5/BI OR 250154-17-7/BI OR 80451-05-4/BI)
L14 2 SEA FILE=HCAPLUS ABB=ON PLU=ON L12 AND L13
L15 4 SEA FILE=HCAPLUS ABB=ON PLU=ON L12 OR L14
L16 509 SEA FILE=HCAPLUS ABB=ON PLU=ON (?PROTEIN? OR ?PEPTID?)(2A)CHI
P
L17 1 SEA FILE=HCAPLUS ABB=ON PLU=ON L16 AND L6
L18 24061 SEA FILE=HCAPLUS ABB=ON PLU=ON (?PROTEIN? OR ?PEPTID?)(5A)(?I
MMOB? OR ATTACH? OR SPAN? OR FIX OR FIXED OR FIXING)
L19 18 SEA FILE=HCAPLUS ABB=ON PLU=ON L6 AND L18
L20 9 SEA FILE=HCAPLUS ABB=ON PLU=ON L19 AND (?ARRAY? OR SURFACE
OR ?SILAN? OR GLASS OR ?MEMBRAN?)
L21 9 SEA FILE=HCAPLUS ABB=ON PLU=ON L20 NOT L15
L22 10 SEA FILE=HCAPLUS ABB=ON PLU=ON L17 OR L21

10 cites

=> d ibib abs 1

L22 ANSWER 1 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2001:126377 HCAPLUS

DOCUMENT NUMBER: 134:293189

TITLE: Biosynthesis of surfactant protein C: characterization of aggresome formation by EGFP chimeras containing propeptide mutants lacking conserved cysteine residues

AUTHOR(S): Kabore, Albert F.; Wang, Wen-Jing; Russo, Scott J.; Beers, Michael F.

CORPORATE SOURCE: Lung Epithelial Cell Biology Laboratories, Pulmonary and Critical Care Division, Department of Medicine, University of Pennsylvania School of Medicine, Philadelphia, PA, 19104, USA

SOURCE: Journal of Cell Science (2001), 114(2), 293-302
CODEN: JNCSAI; ISSN: 0021-9533

PUBLISHER: Company of Biologists Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Surfactant protein C (SP-C) is a lung-specific secreted protein, which is synthesized as a 21-kDa propeptide (SP-C21) and then proteolytically processed as a bitopic **transmembrane** protein in subcellular compartments distal to the medial Golgi to produce a 3.7 kDa mature form. We have shown that initial processing of SP-C21 involves two endoproteolytic cleavages of the C terminus and that truncation of nine amino acids from the C-flanking peptide resulted in retention of mutant protein in proximal compartments. Because these truncations involved removal of a conserved cysteine residue (Cys186), we hypothesized that intraluminal disulfide-mediated folding of the C terminus of SP-C21 is required for intracellular trafficking. To test this, cDNA constructs encoding heterologous fusion proteins consisting of enhanced green fluorescent **protein** (EGFP) **attached** to the N terminus of wild-type rat proSP-C (EGFP/SP-C1-194), C-terminally deleted proSP-C (EGFP/SP-C1-185; EGFP/SP-C1-191) or point mutations of conserved cysteine residues (EGFP/SP-CC122G; EGFP/SP-CC186G; or EGFP/SP-CC122/186G) were transfected into A549 cells. Fluorescence microscopy revealed that transfected EGFP/SP-C1-194 and EGFP/SP-C1-191 were expressed in a punctate pattern within CD-63 pos., EEA-1 neg. cytoplasmic vesicles. In contrast, EGFP/SP-C1-185, EGFP/SP-CC122G, EGFP/SP-CC186G and EGFP/SP-CC122/186G were expressed but retained in a juxtanuclear compartment that stained for ubiquitin and that contained .gamma.-tubulin and vimentin, consistent with expression in aggresomes. Treatment of cells transfected with mutant proSP-C with the proteasome inhibitor lactacystine enhanced aggresome formation, which could be blocked by coincubation with nocodazole. Western blots using a GFP antibody detected a single form in lysates of cells transfected with EGFP/SP-C cysteine mutants, without evidence of smaller degradn. fragments. We conclude that residues Cys122 and Cys186 of proSP-C are required for proper post-translational trafficking. Mutation or deletion of one or both of these residues results in misfolding with mistargeting of unprocessed mutant protein, leading to formation of stable aggregates within aggresomes.

REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs 2

L22 ANSWER 2 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 2000:621123 HCAPLUS

DOCUMENT NUMBER: 133:314134

TITLE: Method for Fabricating Supported Bilayer Lipid
Membranes on GoldAUTHOR(S): **Lahiri, Joydeep; Kalal, Peter;**
Frutos, Anthony G.; Jonas, Steven J.;
Schaeffler, RobertCORPORATE SOURCE: Biochemistry and Surfaces and Interfaces Core
Technologies Science and Technology Division, Corning
Incorporated, Corning, NY, 14831, USASOURCE: Langmuir (2000), 16(20), 7805-7810
CODEN: LANGD5; ISSN: 0743-7463

PUBLISHER: American Chemical Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The paper describes the synthesis of **surfaces** based on self-assembled monolayers (SAMs) of alkanethiolates on Au that were designed to support the adsorption of bilayer lipid **membranes**, and the feasibility of using these **surfaces** for making high-d. **arrays** (HDAs) of **membranes**. The synthesis involved (i) the formation of SAMs of 16-mercaptohexadecanoic acid (MHA) on Au; (ii) the activation of the carboxylic acid groups to interchain anhydrides; (iii) the treatment of the reactive **surfaces** with Brij-76 (C18H37(OCH2CH2)n-10OH) (1) or Brij-76-amine (2) (C18H37(OCH2CH2)n-10NH2); and (iv) the formation of supported **membranes** by incubation or printing of the lipids. The oligo(ethylene glycol) moiety of the anchor lipid provides a hydrophilic spacer between the **surface** and the adsorbed lipid to enable the incorporation of **membrane-spanning proteins** with extra-**membrane** domains. Data from IR spectroscopy confirmed the coupling of 2 to the **surface** through the formation of peptide bonds. Ellipsometric measurements showed an increase of .apprx.15 .ANG. in the thickness of the SAM after coupling to 2; this observation suggests that .apprx.25% of the carboxylic acid groups of the MHA-SAM are derivatized with Brij groups. The yields for coupling of 1 were .apprx.40% of that obsd. for 2. Expts. using **surface** plasmon resonance (SPR) were consistent with the binding of lipid bilayers to the Brij-derivatized **surface**, although alternative structures for the supported lipids cannot be ruled out; by contrast, SAMs of hexadecanethiolate on Au bind lipid monolayers. Biospecific binding of neutravidin was obsd. on supported **membranes** incorporating biotinylated lipids. HDAs of lipids were made by printing lipids onto the Brij-derivatized **surfaces** using a quill-pin printer. Fluorescence microscopy indicated that the printed lipids remained confined to the printed areas; these observations demonstrate the applicability of using com. HDA printers for generating high-d. **membrane arrays** on the Brij-modified **surfaces**.

REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs 3

L22 ANSWER 3 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:617374 HCAPLUS

TITLE: Novel chemistry for parallel synthesis of microarrays of oligonucleotides and peptides using photogenerated acids.

AUTHOR(S): Gao, Xiaolian; LeProust, Eric M.; Pellois, Jean Philippe; Yu, Peilin; Zhang, Hua; Wang, Wei; Zhou, Xiaochuan

CORPORATE SOURCE: Department of Chemistry, University of Houston, Houston, TX, 77204-5641, USA

SOURCE: Book of Abstracts, 218th ACS National Meeting, New Orleans, Aug. 22-26 (1999), MEDI-003. American Chemical Society: Washington, D. C.
CODEN: 67ZJA5

DOCUMENT TYPE: Conference; Meeting Abstract

LANGUAGE: English

AB We present novel soln. chem. that is suitable for parallel synthesis of biomols. and a broad range of org. mols. completed with simple and efficient operations. The novelty of the chem. described herein lies in its use of photo-generated reagents to affect otherwise conventional chem. The synthesis of oligonucleotides using photo-generated acids has been established (Gao et al., J. 120, 12688-12689). In this presentation, the synthesis of peptides using photo-generated acids and combinatorial screening using the addressable chip technol. will be described. Our method using photo-generated reagents rather than conventional reagents provides an optical switch to allow light-initiation of conventional reactions. This new soln. chem. takes the advantage of well-established synthetic procedures and materials and therefore, promises simple, flexible, high quality and low cost parallel synthesis of microarrays of oligonucleotides, peptides and diverse mol. sequences. Parallel synthesis of microarrays of biomols. on planar solid surfaces is a highly efficient means for generating high-d. biochips such as DNA/RNA- and **peptide-chips**, which are powerful high throughput anal. tools. However, the current technologies have significant drawbacks and are not applicable to a vast majority of research and applied labs. in related fields. Our novel chem. forms foundation for a programmable maskless automated microarray synthesizer that is cost-affordable and tech. manageable by most labs. interested in biochips. The availability of such an instrument would significantly accelerate the processes of gene anal. and development of gene-based applications.

=> d ibib abs 4

L22 ANSWER 4 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:17704 HCAPLUS

DOCUMENT NUMBER: 130:234184

TITLE: A Strategy for the Generation of **Surfaces**
Presenting Ligands for Studies of Binding Based on an
Active Ester as a Common Reactive Intermediate: A
Surface Plasmon Resonance StudyAUTHOR(S): **Lahiri, Joydeep**; Isaacs, Lyle; Tien, Joe;
Whitesides, George M.CORPORATE SOURCE: Department of Chemistry and Chemical Biology, Harvard
University, Cambridge, MA, 02138, USA

SOURCE: Anal. Chem. (1999), 71(4), 777-790

CODEN: ANCHAM; ISSN: 0003-2700

PUBLISHER: American Chemical Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB This paper describes the **immobilization** of ten **proteins** and two low-mol.-wt. ligands on mixed selfassembled monolayers (SAMs) of alkanethiolates on gold generated from the tri(ethylene glycol)-terminated thiol 1 (HS(CH₂)₁₁(OCH₂CH₂)₃OH) (.chi.(1) = 1.0-0.0) and the longer, carboxylic acid-terminated thiol 2 (HS(CH₂)₁₁(OCH₂CH₂)₆OCH₂CO₂H) (.chi.(2) = 0.0-1.0). The immobilization was achieved by a two-step procedure: generation of reactive N-hydroxysuccinimidyl esters from the carboxylic acid groups of 2 in the SAM and coupling of these groups with amines on the protein or ligand. Because this method involves a common reactive intermediate that is easily prepd., it provides a convenient method for attaching ligands to SAMs for studies using **surface** plasmon resonance spectroscopy (and, in principle, other bioanal. methods that use derivatized SAMs on gold, silver, and other **surfaces**). These SAMs were resistant to nonspecific adsorption of proteins having a wide range of mol. wts. and isoelec. points. The pH of the coupling buffer, the concn. of protein, the ionic strength of the soln. of protein, and the mol. wt. of the protein all influenced the amt. of the **protein** that was **immobilized**. For the proteins investigated in detail-carbonic anhydrase and lysozyme-the highest quantities of **immobilized proteins** were obtained when using a low ionic strength soln. at a value of pH approx. one unit below the isoelec. point (pI) of the protein, at a concn. of .apprx.0.5 mg mL⁻¹. Comparisons of the kinetic and thermodyn. consts. describing binding of carbonic anhydrase and vancomycin to immobilized benzenesulfonamide and N-.alpha.-Ac-Lys-D-Ala-D-Ala groups, resp., on mixed SAMs (by methods described in this paper) and in the carboxymethyl dextran matrix of com. available substrates yielded (for these systems) essentially indistinguishable values of K_d, k_{off}, and k_{on}.

REFERENCE COUNT: 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS
RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs 5

L22 ANSWER 5 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1999:7458 HCAPLUS

DOCUMENT NUMBER: 130:179567

TITLE: Site-specific immobilization of Fab' fragments of goat antihuman IgG on quartz **surfaces**

AUTHOR(S): Qian, Weiping; **Fang, Yu**; Song, Zhendong; Liang, Bingjie; Yu, Wei

CORPORATE SOURCE: National Laboratory of Molecular and Biomolecular Electronics, Southeast University, Nanjing, 210096, Peop. Rep. China

SOURCE: Supramol. Sci. (1998), 5(5-6), 701-703

CODEN: SUSCFX; ISSN: 0968-5677

PUBLISHER: Elsevier Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB This paper presents a strategy for immobilizing biomols. onto a solid **surface**. We used the free thiol group directing method to immobilize Fab' fragments to the MPTMS derived quartz substrate, and results of X-ray photoelectron spectroscopic studies on the binding of MTPMS and Fab' fragments on the quartz **surfaces** are reported. We also prepd. a new type of immuno-labeled protein which was the rare earth element labeled antigen (human IgG) to recognize Fab' fragment. Four characteristic peaks has been detd. on the **surface** specifically bound human IgG labeled Tb3+.

REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d ibib abs 6

L22 ANSWER 6 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1997:173428 HCAPLUS

DOCUMENT NUMBER: 126:220777

TITLE: Recognition of [3H]oxytocin-receptor complex using
immobilized antisense peptide

AUTHOR(S): Lu, Fengxian; **Wang, Wei**; Tang, Te

CORPORATE SOURCE: Lab. Physiol. Pathol., Tianjin Med. Univ., Tianjin,
300070, Peop. Rep. China

SOURCE: Zhongguo Bingli Shengli Zazhi (1996), 12(3), 239-243
CODEN: ZBSZEB; ISSN: 1000-4718

PUBLISHER: Jinan Daxue

DOCUMENT TYPE: Journal

LANGUAGE: Chinese

AB An antisense peptide for oxytocin, whose sequence was encoded in an antisense strand of DNA corresponding to the 12 N-terminal residues of bovine oxytocin (OT) and its binding protein, neurophysin I, a region that included the OT sequence at residues 1-9 was prepd. The peptide selectively bound to OT and the solubilized complex of OT with receptor protein in plasma **membranes** from rat mammary gland after parturition. The mol. wt. of the OT receptor protein was 65 kDa. The results suggest this peptide might be used as a tool for affinity chromatog.

=> d ibib abs 7

L22 ANSWER 7 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1992:191866 HCAPLUS

DOCUMENT NUMBER: 116:191866

TITLE: Differential glycosylation and cell **surface**
expression of lysosomal **membrane**glycoproteins in sublines of a human colon cancer
exhibiting distinct metastatic potentialsAUTHOR(S): Saitoh, Osamu; Wang, Wei Chun; Lotan,
Reuben; Fukuda, MinoruCORPORATE SOURCE: Cancer Res. Cent., La Jolla Cancer Res. Found., La
Jolla, CA, 92037, USA

SOURCE: J. Biol. Chem. (1992), 267(8), 5700-11

CODEN: JBCHA3; ISSN: 0021-9258

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Changes in the glycosylation of asparagine-linked oligosaccharides have been shown in various tumor cells, including human colon cancer. Attempts were made to elucidate the difference in Asn-linked oligosaccharides **attached** to lysosomal **membrane glycoproteins** isolated from sublines of human colon carcinoma exhibiting high and low metastatic potentials in nude mice. Lysosomal **membrane** glycoproteins (lamp) 1 and 2 were immunopptd. from the cells after labeling with radioactive sugars, and the glycopeptides prepd. were fractionated by serial lectin affinity chromatog. employing immobilized Con A, Datura stramonium agglutinin, and tomato lectin. Comparison of Asn-linked oligosaccharides from the different colonic carcinoma cells revealed the following features. First, the highly metastatic carcinoma cells express more poly-N-acetyllactosaminyl side chains with branched galactose residues than cells with low metastatic potential. Second, sialylation is more significant in the highly metastatic carcinoma cells than in the poorly metastatic ones. Conversely, N-acetyllactosamine units are less fucosylated in the highly metastatic cells than in poorly metastatic cells. These structural changes were apparently caused by the increase in sialyltransferase and the decrease in .alpha.1.fwdarw.3 fucosyltransferase in the highly metastatic cells. The results also suggest that highly metastatic carcinoma cells express more sialyl Lex structures at the termini of poly-N-acetyllactosaminyl side chains than poorly metastatic carcinoma cells. Further, highly metastatic cells were found to express more lamp-1 and lamp-2 on the cell **surface**. These results were found to be correlated to the increased expression of sialyl Lex structures with high affinity binding of anti-sialyl Lex antibody on highly metastatic cells. Increased expression of sialyl Lex in the poly-N-acetyllactosamines of the cell **surface** may contribute to the metastatic behavior of the cells, assuming that this structure can serve as a better ligand for selectins present on endothelial cells and platelets.

=> d ibib abs 8

L22 ANSWER 8 OF 10 HCAPLUS COPYRIGHT 2002 ACS
 ACCESSION NUMBER: 1992:124233 HCAPLUS
 DOCUMENT NUMBER: 116:124233
 TITLE: Sensor of glucose oxidase immobilized in Bombyx mori
 silk fibroin **membrane**
 AUTHOR(S): Shao, Zhengzhong; **Fang, Yue**; Yu, Tongyin;
 Deng, Jiaqi
 CORPORATE SOURCE: Dep. Mater. Sci., Fudan Univ., Shanghai, 200433, Peop.
 Rep. China
 SOURCE: Gaodeng Xuexiao Huaxue Xuebao (1991), 12(6), 847-8
 CODEN: KTHPDM; ISSN: 0251-0790
 DOCUMENT TYPE: Journal
 LANGUAGE: Chinese
 AB Glucose oxidase (I) can be immobilized in B. mori silk fibroin
membrane due to the structural transition of the silk fibroin
membrane from random coil to antiparallel .beta.-sheet induced by
 MeOH treatment. The glucose-selective sensor was made of this I
 immobilized fibroin **membrane**. The calibration curve, response
 time, recovery, and thermal and storage stability were detd.

=> d ibib abs 9

L22 ANSWER 9 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1991:676542 HCAPLUS

DOCUMENT NUMBER: 115:276542

TITLE: The poly-N-acetyllactosamines **attached** to lysosomal **membrane glycoproteins** are increased by the prolonged association with the Golgi complex

AUTHOR(S): **Wang, Wei Chun**; Lee, Ni; Aoki, Daisuke; Fukuda, Michiko N.; Fukuda, Minoru

CORPORATE SOURCE: La Jolla Cancer Res. Found., Cancer Res. Cent., La Jolla, CA, 92037, USA

SOURCE: J. Biol. Chem. (1991), 266(34), 23185-90
CODEN: JBCHA3; ISSN: 0021-9258

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The poly-N-acetyllactosamines on neutrophils and monocytes have been shown to serve as ligands for various selectins present on endothelial cells and platelets. To understand the reason why only certain glycoproteins can be modified by poly-N-acetyllactosamine, the authors utilized 21.degree. incubation conditions, which had previously been shown to cause the accumulation of glycoproteins at the trans-Golgi. HL-60 cells were labeled with [3H]galactose at 21 or 37.degree. for 6 or 24 h, and the lysosomal **membrane** glycoproteins lamp-1 and lamp-2 were immunopptd. On examn. by SDS-PAGE, each lamp from HL-60 cells incubated at 21.degree. exhibited a much broader, slower migrating band than that isolated from the cells incubated at 37.degree.. The no. of N-glycans contg. poly-N-acetyllactosamine, estd. by their binding to tomato lectin column, increased .apprx.30-50% after incubation at 21.degree., compred to incubation at 37.degree.. The anal. of oligosaccharides released by endo-.beta.-galactosidase digestion demonstrates that the amt. of side chains contg. .gtoreq.3 N-acetyllactosamine repeats increased .apprx.100% after incubation at 21.degree., and methylation anal. confirmed these results. The same anal. and the results obtained by ion-exchange chromatog. also provided evidence that the N-glycans of lamps are sialylated at 21.degree. as much as at 37.degree.. Pulse-chase expts. using [35S]methionine labeling indicated that the time necessary for processing of lamps is much longer at 21.degree. than at 37.degree.. These results therefore indicate that incubation at 21.degree. cause the lamps to reside longer within the Golgi complex, and such longer residence allows lamps to acquire more polylactosaminoglycan. These results also suggest that the time necessary for moving through the Golgi complex is a crit. factor for poly-N-acetyllactosamine formation.

=> d ibib abs 10

L22 ANSWER 10 OF 10 HCAPLUS COPYRIGHT 2002 ACS

ACCESSION NUMBER: 1990:589612 HCAPLUS

DOCUMENT NUMBER: 113:189612

TITLE: Granulocytic differentiation of HL-60 cells is associated with increase of poly-N-acetyllactosamine in Asn-linked oligosaccharides **attached** to human lysosomal **membrane glycoproteins**

AUTHOR(S): Lee, Ni; Wang, Wei Chun; Fukuda, Minoru

CORPORATE SOURCE: Cancer Res. Cent., La Jolla Cancer Res. Found., La Jolla, CA, 92037, USA

SOURCE: J. Biol. Chem. (1990), 265(33), 20476-87

CODEN: JBCHA3; ISSN: 0021-9258

DOCUMENT TYPE: Journal

LANGUAGE: English

AB HL-60 cells were induced to differentiate into granulocytic cells by DMSO, and structures of Asn-linked oligosaccharides **attached** to lysosomal **membrane glycoproteins** (lamp-1 and lamp-2) were elucidated before and after differentiation. Lamp-1 and lamp-2 were immunopptd. from the cells after labeling with radioactive sugars, and glycopeptides were prepd. The structures of glycopeptides obtained after serial lectin-affinity chromatog. were elucidated by endo-.beta.-galactoside and methylation anal. Glycopeptides bound to tomato lectin-Sepharose were tetraantennary oligosaccharides that contain two or three poly-N-acetyllactosaminyl chains, of which one side chain contains three or more N-acetyllactosaminyl repeats, whereas those bound to Datura stramonium agglutinin-Sepharose were tetraantennary oligosaccharides contg. one or two short poly-N-acetyllactosaminyl side chains. Glycopeptides that were not bound to Con A, tomato lectin, or D. stramonium agglutinin were triantennary oligosaccharides with a negligible amt. of poly-N-acetyllactosaminyl side chains. Comparison of Asn-linked oligosaccharides from undifferentiated and differentiated HL-60 cells reveals the following features. First, the no. of Asn-linked oligosaccharides contg. poly-N-acetyllactosaminyl side chains increases dramatically with a concomitant decrease in less complex Asn-linked oligosaccharides after differentiation. Second, the no. of poly-N-acetyllactosaminyl side chains per Asn-linked oligosaccharides increases. These increases in poly-N-acetyllactosamine were assocd. with increased activity of UDP-GlcNAc:.beta.-D-Gal-.beta.1 .fwdarw. 3-N-acetylglucosaminyltransferase extension enzyme, a key enzyme in the formation of poly-N-acetyllactosamines. The increased amt. of poly-N-acetyllactosamine in lamp-1 and lamp-2 resulted in longer half-lives of lamp-1 and lamp-2 in differentiated HL-60 cells. These results suggest strongly that the differentiation of HL-60 cells into more phagocytic cells is assocd. with an increase in the complexity of Asn-linked oligosaccharides **attached** to lysosomal **membrane glycoproteins**, which in turn may play a role in stabilizing lysosomes.